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ORIGINAL ARTICLE

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Effect of enhanced geomagnetic activity on hypothermia and mortality in rats

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Abstract The hypothesis was investigated that variability in the severity of limbic seizure-induced hypothermia in rats was affected by ambient geomagnetic activity. Data were obtained in support of this hypothesis. The depth of the hypothermia was significantly ($P < 0.001$) reduced if the ambient geomagnetic activity exceeded 35 nT to 40 nT. Mortality during the subsequent 5 days was increased when the geomagnetic activity was > 20 nT. The magnitude of the effect was comparable to the difference between exposure to light or to darkness during the 20 h after the induction of limbic seizures.

Key words Geomagnetic activity · Light-dark hypothermia · Limbic seizures · Mortality risk

Introduction

Neural thermoregulatory mechanisms are fundamental to the survival of mammals (MacLean 1990). The pattern of brain damage that follows the induction of limbic seizures by a single systemic injection of lithium and pilocarpine has been shown to interfere specifically with the phylogenetically specific thalamocingulate system of the mammal (Peredery et al. 1992). While attempting to understand the sudden cardiac death that sometimes follows the induction of these limbic seizures, Bureau and Persinger (1993) have physically restrained these rats and injected them with the neuroleptic acepromazine (Atravet). The combination of these three variables: restraint, ongoing limbic seizure activity and acepromazine, resulted in a severe hypothermia that remained for approximately 3 days. Although the rat's core body temperature often decreased to ambient levels (20 to 22°C), most of these animals survived.

Despite the precise adherence to the protocol, there was still conspicuous variability in both the severity of the hypothermia and the mortality of these rats within 5 days after the induction of the seizures. Our previous research had indicated that when global geomagnetic activity exceeded 20 nT, mortality also increased within this population during the subsequent 2 to 3 days (Bureau and Persinger 1992). The following experiments were designed to determine: (1) if geomagnetic activity contributed to the variability, and (2) if the magnitude of the geomagnetic effect was similar to continuous lighting during the first 24 h following the induction of the seizures.

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Methods

Experiment I

The first experiment was conducted between January 1993 and June 1994. A total of 311 male albino rats between the ages of 90 and 140 days of age were used. Before and during the experiment, the rats were maintained in standard cages within a climate controlled room (20–22°C); the light-dark cycle was 12:12 h with the local daylight cycle beginning at 0800 hours. Within the housing area, the intensity of the overhead fluorescent lamps during the day cycle ranged from 800 to 1300 lux (Gossen Panlux electronic photometer) and was a function of linear distance.

As a component of the general research program concerning the neurochemical bases of limbic seizures, each rat had been injected subcutaneously (s.c.) with 3 mEq/kg of lithium (between 1100 and 1200 hours, local time), injected 4 h later (s.c.) with 30 mg/kg of pilocarpine and subsequently restrained within a commercial plastic holder. Within 30 min of the onset of the limbic motor seizures, which required about 30 min to emerge overtly, each rat was injected s.c. with 25 mg/kg of acepromazine in order to minimize mortality. During the subsequent 19 h the rats were maintained in the restraint cages within a temperature controlled room (18–21°C) with the overhead, cool fluorescent lights switched on (500 lux). At the end of this period, the rat's core temperature was determined from an Omega model HH21 microprocessor digital thermometer with a K-type thermocouple or YSI series probe and a model 46 TUC tele-thermometer; the probe was inserted approximately 7–10 cm into the rectum.

Survival time, in days, was recorded for each rat. If the rat survives for more than 5 days, its risk of sudden death has been shown to be extremely low (Bureau and Persinger 1992).

Experiment II

In this experiment, a total of 46 male albino rats were exposed, on six different days, immediately following the induction of the seizures to continuous light ($n = 23$) or dark ($n = 23$). Core body temperature and survival time were recorded according to the procedures established in the first experiment. The standard recovery treatment, primarily the availability in bowls of food mush (composed of water and powered rat chow) was available for all rats. Procedures for both experiments were approved by the University animal care committee and followed the Guidelines of the Canadian council on animal care.

The daily geomagnetic activity for the northern hemisphere was estimated from the average antipodal (aa) values, expressed in nanoTesla (or gamma), which were published in the Geomagnetic Indices Bulletin (National Geophysical Data Center, Solar-Terrestrial Physics Center, 325 Broadway, Boulder, Colorado, 80303, USA). These values were obtained for the day of and for each of the 3 days before and after the induction of the limbic seizures. All analyses involved SPSS software (1995 version). To evaluate the magnitude of the putative effect from geomagnetic activity, eta values (a type of correlation coefficient) were obtained.

Results

The results are of dichotomous (above or below the mean) comparisons of successive increments of 24-h geomagnetic acivity occurring concurrently with the duration of the study. Data were obtained of core body temperatures for rats that had been exposed to the experimental treatments described, and are shown in Table 1. The most significant and largest differences between core body temperatures occurred when the threshold for the ambient geomagnetic activity was set between 35 and 50 nT. Below these values there were no statistically significant differences between the core body temperatures while above these values heterogeneity of variance became statistically significant. There was no statistically significant difference [F (1,44) = 1.11, $P > 0.05$] between the core temperatures of rats that had been exposed to the dark compared to those that had been exposed to the light.

Analyses of variance for the survival time (0 to > 5 days) as a function of successive increments of 10 nT activity of the global geomagnetic field demonstrated that the most significant differences [F (1,309) = 4.09, $P < 0.001$; eta = 0.29] occurred for the values obtained during the 24 h prior to the induction of seizures. Polynomial analyses showed a statistically significant quadratic term [F (1,313) = 14.20, $P < 0.001$]. This non-linear relationship between survival time and the amplitude of the variation in geomagnetic activity during the previous 24 h is shown in Fig. 1. Similar successive increments of global geomagnetic activity during each of the 3 days following the induction of the seizures, or during the second or third day before the induction of the seizures, were not significantly associated with alterations in mortality. However, the rats (Experiment II) which were exposed to the dark immediately following the seizure exhibited much lower mortality [F (1,73) = 5.79, $P < 0.01$; eta = 0.26] relative to the rats that had been exposed to the light.

To determine the strength of the relationship between survival time and core body temperature, intercorrela-

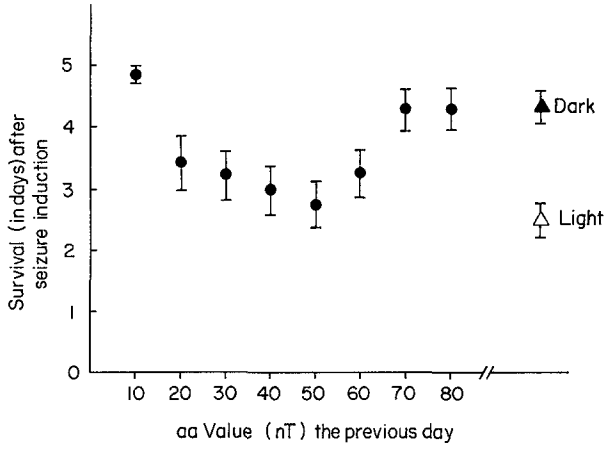

Fig. 1 Means and standard deviations (*vertical bars*) for survival time following the induction of lithium/pilocarpine seizures as a function of geomagnetic activity on the day of induction. For comparison, durations of survival for rats exposed to 24 h of continuous light or normal darkness after the induction are shown. aa, Amplitude of the average daily geomagnetic activity in nT

Table 1 Means and standard deviations for core body temperatures obtained 20 h after the induction of limbic seizures below or above different limena of geomagnetic activity expressed as average antipodal (aa) values on the day of the experiment

aa (nT)	Below threshold			Above threshold			eta-value
Threshold	*n*	Mean	SD	*n*	Mean	SD	
15	35	30.9	5.2	227	31.9	5.9	0.05
20	56	32.1	5.0	206	31.6	6.0	0.04
25	88	31.1	5.5	174	32.1	5.9	0.08
30	112	30.7[a]	5.7	150	32.5[c]	5.8	0.16
35	129	30.2[a]	5.7	133	33.2[c]	5.7	0.25
40	154	30.6[a]	5.6	108	33.3[d]	5.8	0.22
45	168	30.6[a]	5.8	94	33.8[d]	5.4	0.26
50	201	31.1[a]	5.8	61	33.8[d]	5.4	0.20
60	222	31.1[a]	5.9	40	35.0[d]	3.9[d]	0.24

[a vs c] $P<.01$
[a vs d] $P<.001$

tions were performed between the three primary variables for those rats that were still alive ($n = 262$) 19 h after the induction of the seizures, i.e., when the core temperature was taken. To minimize the contributions from potential outliers, nonparametric analyses were employed. Survival time during the 5 days subsequent to the induction of the seizure was significantly ($P < 0.01$) and negatively associated with the core body temperature (rho = –0.13). Seizure onset time (mean ± SD, 30.2 ± 9.7 min) was also positively correlated (Spearman rho = 0.22, $P < 0.001$) with body temperature while survival was not significantly associated (Spearman rho = 0.08, $P > 0.05$) with seizure onset time.

Discussion

Discovery of the severity of the hypothermia due to a synergism of cholinergic-induced limbic seizures, physical restraint and the injection of a phenothiazine derivative acepromazine, has focused our interest upon the intrinsic neuropathways that mediate mammalian homeothermy. The neuropathological changes that follow this treatment involve the same thalamocingulate and hypothalamic pathways that MacLean (1990) has argued to be unique to the mammalian brain. Decrements of approximately 5°C, such as that induced by a *n*-methyl D-aspartate receptor, i.e., NMDA blocker (compound MK-801) following glutaminergic stimulation, have been described as 'hypothermia' (Buchan and Pulsinelli 1990). However, the magnitude of the hypothermia that is evoked by the combined treatments of lithium/pilocarpine, physical restraint and acepromazine is more typical of the type of hypothermia that would be expected when the mammalian thermoregulatory system is compromised: the core temperature becomes asymptotic with ambient temperature.

Our observations that enhanced geomagnetic activity, particularly when it exceeded 35 nT, attenuated the hypothermia suggests that even this acute biochemical disequilibrium is vulnerable to some factor or factors occurring when geomagnetic perturbations surpass this threshold. The existence of a threshold is suggested by the optimal statistical significance between the means when the dichotomous (threshold) analyses were set between 30 and 35 nT. At this limen or threshold value at which measures above or below were compared, the effect size was largest and the minimal heterogeneity of variance existed between the dichotomous clusters.

The neurochemical mechanism by which enhanced geomagnetic activity attenuated the hypothermia may involve the melatonin system. The densest concentration of melatonin receptors has been shown to occur in the suprachiasmatic nucleus (SCN) within the anterior hypothalamus; its role in producing oscillations has been well established (Reghunandanan et al. 1993). SCN neurons responsible for the pineal circadian rhythms have been shown to project directly to the parvocellular neurons of the paraventricular nucleus of the hypothalamus (Klein et al. 1983), which contains the epileptogenic neuropeptide corticotrophin releasing factor (CRF). If these neurons were under excitatory control from neurons within the SCN, a decrease in circulating melatonin levels during enhanced geomagnetic activity could decrease the release of CRF and attenuate the magnitude of the limbic seizures. Alternatively, the depressed melatonin levels would allow the consequences of CRF to be greater upon normal metabolic sequences. The subsequent increased release of adrenocorticotrophic hormone (ACTH) and corticosterone, manifested by enhanced glucogenesis, would oppose the hypothermia.

There is clear evidence that experimentally produced magnetic fields, even when applied for 1 h, have significantly reduced nocturnal levels of melatonin (Yaga et al. 1993). Application of experimentally produced magnetic fields as low as 1 μT for longer periods of time, assuming their structure constitutes a rotating-vector (Kato et al. 1994), have been found to attenuate circulating melatonin levels during both the night and day. The following points should be considered (1) white light attenuates circulating melatonin levels; and (2) most of the rats in this study were exposed to continuous light during the 20 h that followed the induction of the seizure. Therefore the attenuation of the hypothermia could have been encouraged by an interaction of both the continuous light and the geomagnetic activity when it surpassed the limen of 35 nT.

Given the present evidence for attenuation of hypothermia by enhanced geomagnetic activity, it is tempting to speculate that this abiotic variable may be directly or indirectly implicated in certain anomalous thermoregulatory patterns reported among hibernators and estivating species. Individual animals, on occasion, are subject to untimely disruptions leading to arousal from winter dormancy and a premature (but usually unsuccessful) return to active, normothermic life. While the occurrence of such mid-winter arousals is well documented, the factor(s) responsible for triggering of such sporadic anomalous events have remained obscure. The finding of the current study, however, supports the possibility that erratic fluxes in geomagnetic activity may indeed constitute a primary cause. In this context, we suggest that future studies into the untimely arousal of hibernators, estivators and other winter-lethargic species should include an assessment of the occurrence and timing of geomagnetic events surrounding such anomalous events.

The occurrence of a very weak although statistically significant correlation between body temperature and mortality (at least in the rats that survived the first 24 h) and the discrepancy between the limen of geomagnetic activity for these effects suggests that they may be affected by different components of the geophysical variable. The threshold for sudden cardiac death, the most likely etiology of mortality during the 5 days that follow the induction of this form of limbic seizure, was about 20 nT. This threshold is similar to our previous results (Bureau and Persinger 1992) involving fewer ($n = 102$) rats and different measurements. This threshold may not

be unique to rats. Sums of planetary K values (K_p), a type of semi-log representation of the magnitude of geomagnetic activity, were translated into daily aa values for the data reported by Malin and Srivastava (1979). The data indicated that daily cardiac disruptions increased when the geomagnetic activity exceeded about 20 nT.

Rats that had been exposed to complete darkness during the first 20 h after the induction of the seizures exhibited significantly fewer deaths than those exposed to continuous light. The equivalent correlation coefficient was similar to that associated with the effects of the range in geomagnetic activity. Suppression of melatonin secretion by either geomagnetic activity or white light has been hypothesized to contribute to a phasic desynchrony of the central amygdaloid and insular pathways that descend to the parasolitary nuclei (Bureau and Persinger 1992); the resultant simultaneous antagonistic stimulation could evoke sudden cardiac death (asystole). From this perspective should be considered the following recent observation: infants succumbing to sudden death also display significantly smaller pineal organs, which may synthesize and release less melatonin as well as altered aminergic-cholinergic synaptic markers within the hypothalamus (Sparks and Hunsaker 1991). Such findings acquire a new significance in the above context.

The contribution of a putatively subtle variable, such as geomagnetic activity, upon neurochemical processes may not be counterintuitive if one assumes that a variation of the Markov process exists within complex systems near catastrophic limits. During these periods, weak stimuli, particularly if their physical parameters are resonant with the critical subsets of the complex system, can interact and mediate significant directional effects. Time, manifested as the duration of the presentation of the weak stimulus, may be the most underestimated variable determining the initiation of the sequence of events resulting in sudden cardiac death; the critical pattern of pulses may only occur once during the period of exposure. The results of this study strongly indicate that ambient geomagnetic activity can affect the core temperature and the viability of rats in which limbic seizures have been induced.

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